Anterior Choroidal Artery Territory Infarcts
Study of Presumed Mechanisms

D. Leys, MD; F. Mounier-Vehier, MD; I. Lavenu, MD; Ph. Rondepierre, MD; J.P. Pruvo, MD

Background and Purpose A previous study suggested that occlusive diseases of small penetrating arteries account for most anterior choroidal artery (AChA) territory infarcts, but half of the patients did not have an echocardiogram. Cases of AChA territory infarcts associated with internal carotid artery stenosis or atrial fibrillation suggest that this hypothesis may be wrong. The aim of this study was to determine the mechanism of 16 nonselected consecutive AChA territory infarcts.

Methods The study population consisted of 8 men and 8 women aged 17 to 89 years. They underwent a computed tomographic scan at the acute stage, Doppler ultrasonography and B-mode echotomography of the cervical arteries, bidimensional transthoracic echocardiography, and cerebral magnetic resonance imaging, replaced by a second computed tomographic scan in 3 patients. Ten patients underwent cerebral angiography. We defined the presumed cause of stroke according to the criteria used in the trial of Org 10172 in acute stroke treatment.

Results The presumed cause of stroke was definite cardioembolism in 4 patients (atrial fibrillation in 2, paradoxical embolism in 1, and left ventricular akinesia in 1); definite large-vessel atherosclerosis in 2; dissection of the internal carotid artery in 2; and definite small-vessel occlusion in 1. Seven patients had a negative diagnostic workup. Six patients had no risk factors for small-vessel occlusion. The AChA was not visible on angiography in 4 patients. One patient had two arterial cutoffs, suggestive of emboli in other cerebral arteries.

Conclusions This study suggests that AChA territory infarcts are rarely related to small-vessel occlusion and therefore require a complete diagnostic workup. (Stroke. 1994;25: 837-842.)

Key Words • cardioembolic stroke • carotid artery diseases • cerebral arteries • occlusion
Clinical Characteristics, Risk Factors for Stroke, Presumed Cause of Stroke, and Associated Vascular Lesions on Magnetic Resonance Imaging or Computed Tomographic Scans In 16 Patients With Anterior Choroidal Artery Territory Infarction

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Sex</th>
<th>Age, y</th>
<th>Duration of Deficit, h</th>
<th>Size of AChA Territory Infarct, mm</th>
<th>Involvement of Mesiotemporal Territory of AChA</th>
<th>Side of AChA Territory Infarct</th>
<th>Hemiplegia or Hemiparesis</th>
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<td>17</td>
<td>&lt;24*</td>
<td>≥15</td>
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<td>&gt;24</td>
<td>&lt;15</td>
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<td>L</td>
<td>FUL</td>
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</table>

AChA indicates anterior choroidal artery; F, face; U, upper limb; L, lower limb; A, aphasia; HN, hemineglect; HA, hemianopia; AF, atrial fibrillation; VP, valvular prosthesis; DL, dyslipidemia; PFO, patent foramen ovale; Str-TIA, previous stroke or transient ischemic attack; HTN, arterial hypertension; DM, diabetes mellitus; ICA st, internal carotid artery (ICA) stenosis of 50% or more of presumed atherosclerotic origin; LVA, left ventricular akinesia; MCA, middle cerebral artery; and ACA, anterior cerebral artery. Diagnostic criteria were those of Adams et al. 30 See text for diagnostic criteria of HTN, DM, stroke, and TIA. Three patients did not undergo magnetic resonance imaging because of claustrophobia (patient 2), early death (patient 4), or presence of a pacemaker (patient 13).

*Five episodes of transient left pure motor hemiplegia lasting <30 minutes within a 12-hour period.

†Birth control pills were stopped 2 months (patient 1) and 30 months (patient 2) before stroke.

‡Dissection of the ICA.

§AF and VP.

‖Paradoxical embolism.

¶Left ventricular akinesia.

*AF

**Defined according to the criteria of Mounier-Vehier et al. 24 Patients with large-vessel atherosclerosis had a narrowing of the lumen of the ICA of ≥50%. Patients 13 and 14 had a narrowing of the lumen of the ICA of 15% and 25%. The remainder had no atheroma of the ICA.

The group comprised 8 men and 8 women aged 17 to 89 years (median age, 52 years). They were examined at admission by a resident in neurology and within 24 hours by a senior neurologist. We performed the following in all patients: (1) standard blood and urine tests, electrocardiography (ECG), chest radiography, and noncontrast computed tomographic (CT) scan within 24 hours; (2) Doppler ultrasonography and B-mode echotomography of the cervical arteries, bidimensional trans-thoracic echocardiography (TTE), and 24-hour ECG within 15 days; (3) a second CT scan between day 8 and day 15 (at day 2 in patient 4, who had a poor neurological state and died before day 8); and (4) cerebral magnetic resonance imaging (MRI) within 6 weeks after stroke onset, except in 3 patients: 1 who had a pacemaker, 1 who died before MRI could be done, and 1 who refused because of claustrophobia. We performed transesophageal echocardiography within 4 weeks after stroke onset in all patients, except in patients 1 and 2, who had a dissection of the ICA and a normal TTE, and in patient 4, whose consciousness was impaired. A search for aortic arch plaques was not systematically made except for all patients aged older than 60 years who were admitted between March 1, 1992, and February 28, 1993, who were included in the French Aortic Plaques in Stroke (FAPS) study. 19 Evidence of aortic arch plaques was not taken into account for this study. We performed cerebral angiography within 14 days after stroke onset in 10 selected patients (within 2 days in 3 of them). A thorough serological and blood examination was performed to exclude syphilis, hypercoagulability, and hyper-
fibrinogenemia in all patients. An investigation for arteritis was not systematically done. We prospectively collected the following data: age (years); sex; presence of hypertension (defined as systolic blood pressure greater than 140 mm Hg or diastolic blood pressure greater than 80 mm Hg or current treatment with antihypertensive drugs); diabetes mellitus (defined as serum glucose level greater than 1.05 g/L or current use of antidiabetic drugs); dyslipidemia (defined as fasting serum level of triglycerides greater than 1.5 g/L or fasting cholesterol serum level higher than 2.5 g/L); previous TIA or stroke (as defined above); previous myocardial infarction with ECG sequelae; presence of echocardiographic abnormalities such as left ventricular hypertrophy or akinesia, mitral valve prolapse, patent foramen ovale, or aneurysm of the interatrial septum; and significant stenosis of the internal carotid arteries, defined as a narrowing of 50% or more of the lumen (or occlusion) as documented by Doppler ultrasonography, B-mode echotomography, or angiography. The presumed causes of stroke were defined according to the criteria used by Adams et al in the Trial of Org 10172 in Acute Stroke Treatment (TOAST).

Computed tomographic scans were done without contrast by means of 5-mm contiguous slices. All CT scans were done on a Siemens Somatom II machine or on an Ebscint 2004 Elite Plus machine. MRIs were done on an MR-max machine (General Electric) with a superconducting magnet operating at a field strength of 0.5 T. We used axial MRI scans with T₁-weighted spin-echo sequences (repetition time, 2 seconds; two-echo time, 30 and 60 milliseconds), and 6-mm slice thickness with a 6-mm gap. Types and size of infarcts were determined on MRI or on the second CT scan in the 3 patients who did not undergo an MRI. The posterior limb of the internal capsule was involved in all patients. We divided patients between those with AChA infarcts limited to the subcortical territory of the AChA and those with involvement of both subcortical and mesiotemporal territories. We compared the etiologic spectrum of AChA infarcts between both groups by means of Fisher's exact test. We also determined whether patients had associated superficial middle cerebral artery (MCA), laterostriate, anterior cerebral artery (ACA), or posterior cerebral artery (PCA) infarcts, all defined according to Damasio's template. The criteria of Mounier-Vehier et al were used to define centrum ovale, watershed, and internal junctional infarcts. We defined silent infarcts according to the criteria of Mounier-Vehier et al.

Results

The results of the study are presented in the Table. Infarcts located in the AChA territory led to transient

<table>
<thead>
<tr>
<th>Hemihypesthesia</th>
<th>Other Deficits</th>
<th>Risk Factors for Stroke</th>
<th>Presumed Cause of Stroke</th>
<th>Associated Vascular Lesions</th>
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<tr>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Other determined cause†</td>
<td>None</td>
</tr>
<tr>
<td>FUL</td>
<td>A</td>
<td>Nonet†</td>
<td>Other determined cause†</td>
<td>None</td>
</tr>
<tr>
<td>None</td>
<td>HN</td>
<td>Nonet†</td>
<td>Undetermined</td>
<td>Ipsilateral MCA (total) and ACA (posterior part) infarct</td>
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<tr>
<td>FUL</td>
<td>None</td>
<td>AF/VP</td>
<td>Cardioembolism§</td>
<td>Ipsilateral infarct &lt;15 mm in the centrum ovale</td>
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<td>None</td>
<td>DL/PFO</td>
<td>Cardioembolism§</td>
<td>Ipsilateral infarct &lt;15 mm in the centrum ovale</td>
</tr>
<tr>
<td>U</td>
<td>HN</td>
<td>HTN/DM/DL/ICA st</td>
<td>Large-vessel atherosclerosis</td>
<td>Contralateral striatal infarct &lt;15 mm; bilateral silent** infarcts &lt;15 mm in the centrum ovale</td>
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<tr>
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<td>None</td>
<td>HTN/DM/DL/LVA</td>
<td>Cardioembolism§</td>
<td>Contralateral striatal infarct &lt;15 mm; bilateral silent** infarcts &lt;15 mm in the centrum ovale</td>
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<tr>
<td>None</td>
<td>None</td>
<td>HTN/DM</td>
<td>Undetermined</td>
<td>Ipsilateral thalamic infarct</td>
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<td>HA/A</td>
<td>HTN/DM/DL/AF</td>
<td>Small-vessel occlusion</td>
<td>Ipsilateral thalamic infarct</td>
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<td>A</td>
<td>HTN</td>
<td>Undetermined</td>
<td>None</td>
</tr>
<tr>
<td>None</td>
<td>None</td>
<td>HTN/Str-TIA/ICA st</td>
<td>Large-vessel atherosclerosis</td>
<td>Ipsilateral thalamic infarct; contralateral striatal silent** infarct &lt;15 mm</td>
</tr>
</tbody>
</table>

sequences (repetition time, 2 seconds; two-echo time, 30 and 60 milliseconds), and 6-mm slice thickness with a 6-mm gap. Types and size of infarcts were determined on MRI or on the second CT scan in the 3 patients who did not undergo an MRI.
neurological deficits in 4 patients; they were smaller than 15 mm in 5 patients and occurred on the left hemisphere in 7 patients and on the right in 9. All patients had hemiparesis, 8 had hemihypesthesia, 5 had aphasia, 3 had hemineglect, and 3 had hemianopia. Three patients had four risk factors for stroke, 1 had three, 6 had two, and 3 had one. Only 3 patients (patients 1, 2, and 3) had no risk factors for stroke: a 25-year-old woman and a 17-year-old girl, both of whom had a dissection of the ICA, and a 32-year-old woman with a negative diagnostic workup; patients 2 and 3 had a 3-month and a 10-year history of using birth control pills, which had been stopped 2 and 19 months before stroke onset, respectively. Six patients had no risk factors for small-vessel occlusion (patients 1 through 6).

The AChA was patent on angiography in 6 patients who had no sign of emboli. The AChA was not visible in 4 patients (patients 4 through 7); angiography was not sensitive enough to detect small arteries such as the AChA in all patients, and a diagnostic cutoff suggestive of emboli at the origin of the MCA and in a branch of the callosomarginal artery. None of the remaining patients who underwent angiography had another intracranial arterial abnormality. We found criteria for cardioembolism in 4 patients: large-vessel atherosclerosis in 2 patients, other determined cause of stroke in 2 patients with dissection of the ICA, and small-vessel occlusion in 1 patient. The presumed causes of stroke remained undetermined in 7 patients who had a negative diagnostic workup. Of the 8 patients with involvement of the subcortical and the mesiotemporal territories of the AChA, 7 had a determined cause of stroke (cardioembolism in 3, large-vessel atherosclerosis in 2, and dissections of the ICA in 2); only 1 patient with an infarct limited to the subcortical territory of the AChA had a determined cause of stroke (small-vessel occlusion in 1 and cardioembolism in 1). This difference is statistically significant (2P=.02). The infarct size did not significantly differ between both groups: of the 8 patients with involvement of the subcortical and the mesiotemporal territories of the AChA, 7 had an infarct of 15 mm or greater; of the 8 patients with an infarct limited to the subcortical territory of the AChA, 4 had an infarct of 15 mm or greater (2P=.49).

Six patients had another infarct: large ipsilateral MCA and ACA infarcts from a cardioembolic source in 1 patient, small infarcts in the centrum ovale in 3 patients, ipsilateral thalamic infarct in 2 patients, and contralateral infarct greater than 15 mm in the striatum in 2 patients (some patients had more than one associated lesion; see the Table). Three associated brain infarcts were silent: two were located in the centrum ovale and one in the striatum. Ten patients had no other brain infarct.

Discussion

This study revealed that of 16 patients with an AChA territory infarct, 9 had risk factors for occlusive disease of penetrating arteries but only 1 fulfilled the criteria for small-vessel occlusion. None of the previous studies on AChA territory infarcts have performed a systematic search for ICA stenosis and possible cardiac source of emboli. In the study of Bruno et al16 only 15 of 31 patients underwent echocardiography, and this may have led to an underestimation of the potential cardiac sources of stroke. A selection bias leading to an overrepresentation of patients with ICA atherosclerosis and cardioembolism in our study group is unlikely because we only included patients admitted as emergency cases within 24 hours after stroke onset. The modality of recruitment in other studies was not always explained, and recruitment bias might explain discrepancies. In the study of Weiller et al25 large striatocapsular infarcts involving the AChA territory were usually related to ICA atherosclerosis; however, their 11 patients with AChA territory infarcts also had infarcts in the territory of the deep perforators of the MCA, and this may have skewed the results to an overrepresentation of large-vessel diseases in their study population. Our study group was similar to most published cases of AChA territory infarcts from clinical and radiological points of view and may therefore be considered representative of patients with such infarcts.2,16

The AChA was patent on angiography in 6 patients: they had no angiographic sign of emboli in the AChA or in other cerebral arteries. Helgason et al13 found a narrowing of the petrous ICA in a patient with AChA territory infarct, but they did not mention the degree of stenosis. Of 17 patients with AChA territory infarcts who underwent an angiography, Bruno et al16 found only 1 patient with mild stenosis of the carotid siphon and 2 with a nonvisible AChA. A nonvisible AChA was more frequent in our study (4 of 10); this difference may be due to the delay between stroke onset and angiography, which was not mentioned in Bruno's study.16 However, a nonvisibility of the AChA, present in 5% of all angiograms,26 does not always mean its proximal occlusion. In patient 4 with cardioembolism we found 2 emboli in other cerebral arteries and a nonvisibility of the AChA that might have been due to its occlusion at origin. However, an extension of the occlusion to the branches of the AChA is probable16; an occlusion of the origin of the AChA does not lead to neurological deficits27,28 because of anastomoses via the posterior communicating artery and PCA.29 Occlusions of the branches of the AChA cannot be diagnosed with certainty on angiography because of their small size.16 In previous studies, as in ours, an angiography was done only in selected patients; although we performed angiograms during a few days after stroke onset, angiographic signs of emboli may have disappeared. Noninvasive techniques such as magnetic resonance angiography will probably allow the systematic investigation of the intracranial arteries in all patients with AChA territory infarcts.30 In the near future it may be a valuable tool to detect emboli before a spontaneous recanalization occurs, providing that it is done soon after stroke onset. However, at present this technique is not sensitive enough to detect small arteries such as the AChA in all cases.

The number of patients with cardiac sources of emboli (4 of 16) was higher in our study than in previous series of patients with AChA territory infarcts: 2 of 31 for Bruno et al16 1 of 16 for Decroix et al,7 5 of 27 for Sterbini et al31 and 2 of 11 for Helgason et al.32,33 The higher incidence of possible cardiac sources of emboli in our study may be due to the more extensive diagnostic workup.

Four of 15 patients had an extracranial ICA stenosis greater than 50% or occlusion. Coexistence of small-vessel occlusion cannot be excluded in our 2 patients
with ICA atherosclerosis and in similar cases in the literaturebecause arterial hypertension is the major risk factor for most causes of cerebral infarcts. However, patients 1 and 2 were both young patients who had a definite cause of stroke and no risk factors for stroke; they are therefore unlikely to have concomitant small-vessel occlusion. Bruno et al. found only 4 of 31 patients with an AChA territory infarct who had an ICA stenosis, including only 2 with a narrowing of the lumen of 50% or more. Of 27 patients with AChA territory infarction, Sterbini et al. found only 3 patients with ICA stenosis, but they did not mention the degree of stenosis; this very low percentage of significant ICA stenosis was similar to that reported in asymptomatic individuals. Of 6 patients with an AChA territory infarct, Helgason et al. found 1 patient with mild bilateral stenosis of the ICA. The number of ICA stenoses or occlusions was higher in our study and in the study of Decroix et al., in which 5 of 13 patients with an AChA territory infarct had a so-called moderate stenosis of the ICA. The possible role of ICA stenosis in the pathogenesis of subcortical infarcts may also be inferred from the 8% rate of small deep infarcts found by Ghika et al. in patients with severe stenosis or occlusion of the ICA, half of whom had no risk factors for small-vessel occlusion.

Only 1 of 16 patients fulfilled the criteria for small-vessel occlusion. However, the criteria of Adams et al. for small-vessel occlusion require (1) a lacunar syndrome; (2) the absence of hemianopia, hemineglect, and aphasia; (3) the absence of cerebral infarction of 15 mm or greater; (4) the absence of potential cardiac cause of infarction; and (5) the absence of extracranial artery stenosis greater than 50%. These criteria are very restrictive and probably lead to an underestimation of the prevalence of small-vessel occlusion. Therefore, we cannot preclude that some infarcts of so-called undetermined cause may also be the consequence of small-vessel occlusion, especially in patients with arterial hypertension or diabetes mellitus and in the elderly. However, this seems unlikely in our patients with undetermined causes of stroke who had either an infarct larger than 15 mm (patients 3, 6, 7, and 14) or hemianopia (patient 15), hemineglect (patients 3 and 9), or aphasia (patients 14 and 15). The occlusion of a single perforator usually leads to infarcts smaller than 15 mm, and patients usually remain without hemianopia, hemineglect, or aphasia. Moreover, 6 patients had no risk factors for small-vessel occlusion, and we found a definite cause of infarction different from small-vessel occlusion in 8 patients. These findings suggest that small-vessel occlusion accounts for only 1 of 16 AChA territory infarcts, or possibly 2 of 16 if we classify in the “small-vessel occlusion” group patient 11, who had a pure motor stroke sparing the lower limb and an infarct smaller than 15 mm. However, risk factors for small-vessel occlusion may have contributed to AChA territory infarct in 9 other patients. These data lead to the conclusion that at least one third of AChA territory infarcts cannot be the consequence of a small-vessel occlusive disease. Bruno et al. found a low rate of carotid artery stenosis and potential cardiac sources of emboli in similar patients and inferred that small-vessel occlusion is probably the main cause of AChA territory infarcts. However, their main criterion for small-vessel occlusion was the exclusion of other causes. They did not take into account the clinical pattern (presence of a lacunar syndrome) and the size of the infarct.

Our study also showed that most patients who have an infarct involving both the subcortical and mesiotemporal territories of the AChA have a definite cause of stroke different from small-vessel occlusion; therefore, small-vessel occlusion is unlikely to be the presumed mechanism of stroke in these patients.

We conclude that most AChA territory infarcts are not due to small-vessel occlusion, especially in patients with involvement of both the subcortical and mesiotemporal territories of the AChA. Therefore, a complete diagnostic workup remains necessary in all patients with AChA territory infarcts.

Acknowledgments

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